Amyotrophic Lateral Sclerosis (ALS) is a rapidly progressing fatal neurodegenerative disorder. Deterioration of motor neurons results in the loss of voluntary movement control and eventually breathing; with a median life expectancy of three to five years post diagnosis. Traditional drug development, focused on pathological endpoints, has failed to deliver meaningful interventions to halt the progression of neurodegenerative disease.

By targeting fundamental cellular processes, such RNA-Protein interactions, we are leading the discovery of a novel class of targets with preclinical efficacy across a number of neurodegenerative diseases.

ALS, like other neurodegenerative diseases, is associated with the emergence of multiple inflammatory stressors that set neurons on a path to premature cellular death known as necroptosis. By targeting a complex protein-protein interface, we are able to halt necroptosis without interfering with normal homeostatic-apoptotic pathways. Leveraging CIBS infrastructure and expertise we are advancing the preclinical testing required to move this compound forward.

**PRE-EXISTING AUTOIMMUNE DISORDERS HAVE BEEN IMPLICATED IN THE PATHOGENESIS OF ALS.**

We are pioneering the development of patient specific nucleic-acid aptamers capable of neutralizing pathological antibodies in neurodegenerative diseases where cellular damage is caused by an autoimmune response.

Further, by virtually screening compounds with demonstrated efficacy in one disease, we are discovering common pathways with other age-associated diseases; Alzheimer’s Disease, Parkinson’s and Multiple Sclerosis.